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| HXB2 Location | Author's Location Sequence | ence | Immunogen | Species (HLA) | References |
|---------------|---|---|---|--|--|
| | • Among HIV+ individuals who carried HLA B35, 4/21 (19%) recognized this epitope. | arried HLA B35, 4/21 (19 | 9%) recognized this epitope. | | |
| RT (175–183) | Pol Keywords mother-to-infant transmission. Donor HLA A3, A11, B35, B51. • IFNgamma T-cell responses in breast milk of 5 HIV-1 infected women from responded to Gag, 8/11 responded to Pol, 7/11 women to Nef, and 2/5 wome CD8+ T-cells in one woman, and another woman had cytolytic responses mea • T-cells in breast milk from a volunteer who was HLA A3, A11, B35, B51 in epitope NPDIVIYQY. • The frequencies of responses in the two compartments differed, and 2/4 wo responses in breast milk but no detectable responses in peripheral blood cells. | smission. 1. breast milk of 5 HIV-1 in led to Pol, 7/11 women to d another woman had cytulunteer who was HLA A of the two compartments of detectable responses in pedicinal simple. | Keywords mother-to-infant transmission. Donor HLA A3, A11, B35, B51. ENgamma T-cell responses in breast milk of 5 HIV-1 infected women from the US and 6 from Zambia were tested with using Elispot. 11/11 women responded to Gag, 8/11 responded to Pol, 7/11 women to Nef, and 2/5 women to Env peptide pools. These responses were shown to be primarily due to CD8+ T-cells in one woman, and another woman had cytolytic responses measured by Cr-release. T-cells in breast milk from a volunteer who was HLA A3, A11, B35, B51 induced IFNgamma after stimulation with a peptide that carries known B35 epitope NPDIVIYQY. The frequencies of responses in the two compartments differed, and 2/4 women that responded to epitopes in Nef 101-205 and Pol 601-710 showed responses in breast milk but no detectable responses in peripheral blood cells. | human (B35) from Zambia were tested w le pools. These responses v ease. ma after stimulation with a nded to epitopes in Nef 10 | Sabbaj2002a with using Elispot. 11/11 women were shown to be primarily due to a peptide that carries known B35 01-205 and Pol 601-710 showed |
| RT (175–183) | Vaccine Vector/Iype: DNA prime with modified vaccinia Ankara (MVA) boost Keywords inter-clade comparisons, epitope processing, vaccine-specific epitope The HIV-1 subtype A focused vaccine HIVA contains p24 and p17, in a reversed- could direct the protein to the cell membrane and inhibit efficient peptide proces often immunodominant epitopes that were selected to have particularly good cro and MVA prime-boost vaccination protocol using the HIVA antigen will be use polyepitope string [Hanke2000]. Multiple CD4+ or CD8+ T-cell vaccine-induced responses to peptide pools were assays after vaccination of 5 macaques. The response to the Mamu A*01 SIV p; not immunodominant in the Mamu A*01 vaccinated macaques, possibly because [Wee2002]. | NIYQY ne with modified vaccinia ons, epitope processing, vaccine HIVA contains p24 ell membrane and inhibit s that were selected to have ion protocol using the HI vaccine-induced respons caques. The response to turn A*01 vaccinated mac | | ine human, macaque (B35) Hanke2000, Strain: A clade HIV component: p17 Gag, p24 Gag characteristics, immunodominance. order relative to the Gag polyprotein to prevent myristy sing and class I presentation, as well as a polyepitope is ss-reactive potential for the A-clade epidemic in Nairc din a phase III clinical trial in Kenya. This epitope e detected using intracellular cytokine staining and II! ?? epitope p11C (CTPYDINQM), included in the poly e of processing limitations in context of the artificial | Hanke2000, Wee2002 7 Gag, p24 Gag prevent myristylation of p17, which a polyepitope string of conserved, idemic in Nairobi, Kenya. A DNA This epitope is included in the staining and IFNgamma Elispot ded in the polyepitope region, was of the artificial polyepitope string |
| RT (175–184) | RT (175–184 LAI) NPDIVIYQYM • This epitope contains the mutation M184V, a freque • Patient 246#1 (B51), was found by ELISPOT to rec • The resistance mutation M184V gave an increased gequence and also an increased ELISPOT reactivity. | IVIYQYM on M184V, a frequent mu by ELISPOT to recognize gave an increased predict ELISPOT reactivity. | RT (175–184 LAI) NPDIVIXQYM HIV-1 infection human (B51) Samri2000 This epitope contains the mutation M184V, a frequent mutation induced by nucleoside reverse transcriptase inhibitors. Patient 246#1 (B51), was found by ELISPOT to recognize the wild type and the mutated peptide after zidovudine treatment. The resistance mutation M184V gave an increased predicted binding score to B51 (http://bimas.dcrt.nih.gov/molbio/hla_bind) compared to the wildtype RT sequence and also an increased ELISPOT reactivity. | human (B51) e transcriptase inhibitors. tide after zidovudine treatm as.dcrt.nih.gov/molbio/hla_ | Samri2000 tent. bind) compared to the wildtype RT |
| RT (175–199) | RT (342–366 LAI) NPDIVIYQYMDDLYVGSDL- HIV-1 infe E1GQHR • One of five epitopes defined for RT-specific CTL clones in this study. | NPDIVIYQYMDDLYVGSDL- EIGQHR ed for RT-specific CTL clones in | HIV-1 infection n this study. | human (A11) | Menendez-Arias1998, Walker1989 |
| RT (179–187) | RT VIYQYMDDL Vaccine Vector/Type: vaccinia This epitope was shown to be processed and presere carrying 20 HIV-1 epitopes recognized by humans. | VIYQYMDDL inia be processed and presented to s recognized by humans. | RT Vaccine human (A*0201) Hanke1998a, Hanke1998b Vaccine Vaccine human (A*0201) Hanke1998a, Hanke1998b vaccinia This epitope was shown to be processed and presented to appropriate CTL clones upon infection of human target cells with vaccinia virus Ankara (VVA) carrying 20 HIV-1 epitopes recognized by humans. | human (A*0201) ction of human target cells v | Hanke1998a, Hanke1998b with vaccinia virus Ankara (VVA) |
| RT (179–187) | RT VIYOYM • Adoptive transfer of two autologous into a patient – they were well toler treatment had no impact upon viral | VIYQYMDDL utologous in vitro-expanded CTL clones aga well tolerated, but the SLYNTVATL clone v pon viral load and CD4 and CD8 cell counts. | RT Adoptive transfer of two autologous <i>in vitro</i> -expanded CTL clones against the A*0201 restricted epitopes SLYNTVATL and VIYQYMDDL were infused into a patient – they were well tolerated, but the SLYNTVATL clone was shown by tetramer staining to be rapidly eliminated through apoptosis, and the treatment had no impact upon viral load and CD4 and CD8 cell counts. | human (A*0201) cted epitopes SLYNTVATL r staining to be rapidly elim | Tan1999 Land VIYQYMDDL were infused innated through apoptosis, and the |

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| | Tetramer staining failed for the VIY | d for the VIYQYMDDL epit | QYMDDL epitope as the tetramer was unstable. | | |
| RT (179–187) | Pol (346–354) Keywords epitope proo Proteasome regulation The proteasome is inhi the proteasome to creat IFN-gamma induction the presentation of the pathways. ILKEPVHGV seems to This epitope contains to | Keywords epitope processing, immunodominance, escape. Proteasome regulation influences epitope processing and co. The proteasome is inhibited by lactacystin treatment, and g the proteasome to create an immunoproteasome. IEN gamma induction of the immunoproteasome and lacta the presentation of the A*0201 ILKEPVHGV epitope, whi pathways. ILKEPVHGV seems to be processed by the classical protest This epitope contains the catalytic site (YMDD) of RT, a co. | Pol (346–354) VIYQYMDDL HIV-1 infection human (A*0201) Sewell1999 Keywords epitope processing, immunodominance, escape. Proteasome regulation influences epitope processing and could influence patterns of immunodominance. The proteasome is inhibited by lactacystin treatment, and gamma IFN induces expression of proteasome subunits, LMP2 and LMP7, which combine with the proteasome to create an immunoproteasome. IFN-gamma induction of the immunoproteasome and lactacystin inhibition increases the presentation of the A*0201 ILKEPVHGV epitope, which is immunodominant within pol proteins, showing the two epitopes are processed by different pathways. ILKEPVHGV seems to be processed by the classical proteasome pathway, while VIYQYMDDL appears to be destroyed by this pathway. This epitope contains the catalytic site (YMDD) of RT, a conserved sequence in HIV-1 which restricts escape mutants. | human (A*0201) unodominance. n of proteasome subunits, LM presentation of the A*0201 V pol proteins, showing the tw (MDDL appears to be destroy-hich restricts escape mutants. | Sewell1999 IP2 and LMP7, which combine with TYQYMDDL epitope, but decreases o epitopes are processed by different ed by this pathway. |
| RT (179–187) | RT (346–354 LAI) VIYQYM Keywords review. The substitution VIYQYVDDL abra [Menendez-Arias1998], in a review, | VIYQYMDDL YYVDDL abrogates CTL resp. I, in a review, notes that this e | RT (346–354 LAI) VIYQYMDDL HIV-1 infection human (A*0201) Harrer1996 Keywords review. The substitution VIYQYVDDL abrogates CTL response and confers drug resistance. [Menendez-Arias1998], in a review, notes that this epitope includes catalytic residues (Asp-185 and Asp-186) in the active site of RT. | human (A*0201) sp-185 and Asp-186) in the ac | Harrer1996a, Menendez-Arias1998 tive site of RT. |
| RT (179–187) | RT $(346-354 LAI)$ VIYQYM • C. Brander notes this is an $A*0201$ | VIYQYMDDL s an A*0201 epitope. | HIV-1 infection | human (A*0201) | Frahm2004 |
| RT (179–187) | RT (346–354) | VIYQYMDDL | HIV-1 infection | human (A*0201) | Brander1998a, Menendez-Arias1998 |
| | • Of 17 infected HLA A*0201 subjects, 13 VIYQYMDDL, and there was no correlation only one subject had CTL against all three subjects were part of the San Francisco Cirol the review [Menendez-Arias1998] the a substitutions VIE and M6V abolish CTI resistance to non-nucleoside RT inhibitors. | Neywords review, escape. Of 17 infected HLA A*0201 subjects, 13 had CTL VIYQYMDDL, and there was no correlation betwee Only one subject had CTL against all three epitopes. Subjects were part of the San Francisco City Clinic (In the review [Menendez-Arias1998] the authors no – substitutions VIE and M6V abolish CTL activity, resistance to non-nucleoside RT inhibitors. | NEWWORDS TEVEN, escape. Of 17 infected HLA A*0201 subjects, 13 had CTL responses against the p17 SLYNTVATL epitope, six recognized ILKEPVHGV and five recognized VIYQYMDDL, and there was no correlation between viral load and recognition of a specific epitope or evidence of immune escape. Only one subject had CTL against all three epitopes. Subjects were part of the San Francisco City Clinic Cohort, the ARIEL project and from the Boston area. In the review [Menendez-Arias1998] the authors note that substitution of three residues in this epitope can confer resistance to RT inhibitors (1, 3, and 6) – substitutions V1E and M6V abolish CTL activity, and M6V confers resistance to 3TC – substitution Y3C reduces CTL activity and is associated with resistance to non-nucleoside RT inhibitors. | ATL epitope, six recognized cific epitope or evidence of in the Boston area. in this epitope can confer resi : - substitution Y3C reduces (| ILKEPVHGV and five recognized umune escape. stance to RT inhibitors (1, 3, and 6) CTL activity and is associated with |
| RT (179–187) | KEywords inter-clade comparisons, Epitope name RT VL9. • HIV was scanned for all peptides whand 30 of these bound to HLA-A*0. • Three additional previously describe had CTL that recognized at least one of 1 and maximum of 2). • RT VL9 was not recognized by any in this study. | VIYQYMDDL comparisons, supertype, com 9. Ill peptides which carried the 4 to HLA-A*0201 – 20/30 bou ously described HLA-A2 epit ed at least one of the 23 peptia 2) mized by any of the 22 HLA-4 mized by any of the 22 HLA-4 | Keywords inter-clade comparisons, supertype, computational epitope prediction. Epitope name RT VL9. HIV-1 infection. Epitope name RT VL9. HIV was scanned for all peptides which carried the A2-supermotif pattern conserved in more than 50% of B clade sequences – 233 peptides met this criteria, and 30 of these bound to HLA-A*0201 – 20/30 bound to at least 3/5 of HLA-A2 supertype alleles tested. Three additional previously described HLA-A2 epitopes were added to the set of 20, including RT VL9, and 18/22 chronically infected HLA-A2 individuals had CTL that recognized at least one of the 23 peptides (median of 2 and maximum of 6), while 6/12 acute infected individuals recognized at least 1 (median of 1 and maximum of 2) RT VL9 was not recognized by any of the 22 HLA-A2 patients with chronic HIV-1 infection or the 13 HLA-A2 patients with acute HIV-1 infection included in this study. | human (A*0201) ore than 50% of B clade seque pe alleles tested. uding RT VL9, and 18/22 chro while 6/12 acute infected indi ion or the 13 HLA-A2 patients | Altfeld2001c ences – 233 peptides met this criteria, nically infected HLA-A2 individuals recognized at least 1 (median s with acute HIV-1 infection included |

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| RT (179–187) | RT (346–354) Epitope name VL9. Integration of HIV RT Clysis by CD8+ CTL. | RT (346–354) VIYQYMDDL HIV-1 infection Epitope name VL9. • Integration of HIV RT CTL epitopes into the N-terminus of the HLA-A2 h lysis by CD8+ CTL. • These antigens could also be used to stimulate primary responses <i>in vitro</i> . | RT (346–354) VIYQYMDDL HIV-1 infection human (A*0201) Dela Cruz 2000 Epitope name VL9. • Integration of HIV RT CTL epitopes into the N-terminus of the HLA-A2 heavy chain, or tethering the epitopes to the target chain, resulted in epitope-specific lysis by CD8+ CTL. • These antigens could also be used to stimulate primary responses in vitro. | human (A*0201) tering the epitopes to the tar | Dela Cruz2000 get chain, resulted in epitope-specific |
| RT (179–187) | Pol (346–354) VIYQYM Keywords epitope processing, imm • Epitope processing of three differen 1.74 cells were used that lack TAP1 genes could be added back through • ILKEPVHGV was efficiently prese by the MB1 subunit of the protease restored. SLYNTVATL expression | Fol (346–354) VIYQYMDDL Keywords epitope processing, immunodominance. Epitope processing of three different HLA-A*0201 1.74 cells were used that lack TAP1 and TAP2 gene genes could be added back through transfection to sILKEPVHGV was efficiently presented in TAP-1 a by the MB1 subunit of the protease, and could be erestored. SLYNTVATL expression was unaltered by | Pol (346–354) VIYQYMDDL HIV-1 infection human (A*0201) Sewell2002 Keywords epitope processing, immunodominance. • Epitope processing of three different HLA-A*0201 HIV epitopes was shown to use different pathways, which might influence patterns of immunodominance. 174 cells were used that lack TAP1 and TAP2 genes, as well as the LMP2 and LMP7 genes that encode the beta-subunits of the immunoproteasome. These genes could be added back through transfection to study processing. • ILKEPVHGV was efficiently presented in TAP-1 and -2 transfected cells while VIYQYMDDL and SLYNTVATL were not. VIYQYMDDL was destroyed by the MB1 subunit of the protease, and could be expressed in the presence of the proteasome inhibitor lactacystin, but SLYNTVATL expression was not restored. SLYNTVATL expression was unaltered by lactacystin in a wild type cell line. | human (A*0201) pathways, which might infl: that encode the beta-subul DL and SLYNTVATL were inhibitor lactacystin, b | Sewell2002 uence patterns of immunodominance. iits of the immunoproteasome. These ie not. VIYQYMDDL was destroyed ut SLYNTVATL expression was not |
| RT (179–187) | Vaccine Vector/Type: DNA prime v Keywords inter-clade comparisons. The HIV-1 subtype A focused vacci could direct the protein to the cell roften immunodominant epitopes the and MVA prime-boost vaccination polyepitope string [Hanke2000]. Multiple CD4+ or CD8+ T-cell vac assays after vaccination of 5 macaq not immunodominant in the Mamu [Wee2002]. | VIYQYMDDL NA prime with modified v omparisons, epitope procescused vaccine HIVA contato the cell membrane and epitopes that were selected vaccination protocol using ke2000]. H. T.cell vaccine-induced of 5 macaques. The respont the Mamu A*01 vaccinary | Pol VIYQYMDDL HIV-1 infection, Vaccine human, macaque Hanke2000, Wee2002 (A*0201) Vaccine Vector/Type: DNA prime with modified vaccinia Ankara (MVA) boost Strain: A clade HIV component: p17 Gag, p24 Gag Keywords inter-clade comparisons, epitope processing, vaccine-specific epitope characteristics, immunodominance. • The HIV-1 subtype A focused vaccine HIVA contains p24 and p17, in a reversed order relative to the Gag polyprotein to prevent myristylation of p17, which could direct the protein to the cell membrane and inhibit efficient peptide processing and class I presentation, as well as a polyepitope string of conserved, often immunodominant epitopes that were selected to have particularly good cross-reactive potential for the A-clade epidemic in Nairobi, Kenya. A DNA and MVA prime-boost vaccination protocol using the HIVA antigen will be used in a phase III clinical trial in Kenya. This epitope is included in the polyepitope string [Hanke2000]. • Multiple CD4+ or CD8+ T-cell vaccine-induced responses to peptide pools were detected using intracellular cytokine staining and IFNgamma Elispot assays after vaccination of 5 macaques. The response to the Mamu A*01 SIV p27 epitope p11C (CTPYDINQM), included in the polyepitope region, was not immunodominant in the Mamu A*01 vaccinated macaques, possibly because of processing limitations in context of the artificial polyepitope string [Wee2002]. | human, macaque (A*0201) clade HIV component: p stics, immunodominance. ve to the Gag polyprotein t ass I presentation, as well potential for the A-clade e se III clinical trial in Keny using intracellular cytokii p11C (CTPYDINQM), inc | Hanke2000, Wee2002 17 Gag, p24 Gag o prevent myristylation of p17, which as a polyepitope string of conserved, pidemic in Nairobi, Kenya. A DNA a. This epitope is included in the estaining and IFNgamma Elispot luded in the polyepitope region, was t of the artificial polyepitope string |
| RT (179–187) | RT (179–187) VIYQYM Vaccine Vector/Type: peptide HIN Keywords binding affinity, vaccine Assay type cytokine production, CI Donor HLA A2.1. • Alanine substitutions of VIYQYMI fold higher MHC binding affinity the higher affinity form of vLyqymddV | KT (179–187) VIYQYMDDL Vaccine Vector/Type: peptide HIV component: RT Adj Keywords binding affinity, vaccine-induced epitopes. Assay type cytokine production, Chromium-release assay. Donor HLA A2.1. Alanine substitutions of VIYQYMDDL were tested for im fold higher MHC binding affinity than wild type. YLygym higher affinity form of vLygymddV induced CTL in vivo t | RT (179–187) VIYQYMDDL Vaccine mouse (A*0201) Okazaki2003 Vaccine Vector/Type: peptide HIV component: RT Adjuvant: Incomplete Freund's Adjuvant (IFA), IL-12 Keywords binding affinity, vaccine-induced epitopes. Assay type cytokine production, Chromium-release assay. Donor HLA A2.1. Alanine substitutions of VIYQYMDDL were tested for importance of each amino acid for HLA-A2.1 binding. Peptide variant (vLyqymddV) showed an 8 fold higher MHC binding affinity than wild type. YLyqymddV had an even higher binding affinity, but the Y at positions one blocked TCR recognition. The higher affinity form of vLyqymddV induced CTL in vivo that could protect against a vaccinia virus expressing RT and the wild type epitope. | mouse (A*0201) rvant (IFA), IL-12 HLA-A2.1 binding. Peptid uffinity, but the Y at positio ia virus expressing RT and | Okazaki2003 e variant (vLyqymddV) showed an 8 ns one blocked TCR recognition. The the wild type epitope. |
| RT (179–187) | KEY VIYOYM Keywords inter-clade comparisons A CTL response was found in expo and D clades – such cross-reactivity The A and D consensus sequences: | KT Keywords inter-clade comparisons. A CTL response was found in exposed but uninfected prost and D clades – such cross-reactivity could protect against be The A and D consensus sequences are both VIYQYMMDL | KEY VIYQYMMDL HIV-1 exposed seronegative human (A2) Rowland-Jones1998a Keywords inter-clade comparisons. A CTL response was found in exposed but uninfected prostitutes from Nairobi using previously-defined B clade epitopes that tended to be conserved in A and D clades – such cross-reactivity could protect against both A and D and confer protection in Nairobi where both subtypes are circulating. The A and D consensus sequences are both VIYQYMMDL. | human (A2) ously-defined B clade epito on in Nairobi where both sı | Rowland-Jones1998a pes that tended to be conserved in A abtypes are circulating. |

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| RT (179–187) | Pol (346–354) VIYOYI Vaccine Vector/Type: DNA prime A polyepitope vaccine was general HHD mice have a transgene of HL in the mice. CTL responses to Gag (77-85) SI observed in HIV polytope HHD-va No CTL immune responses were a Nef 180-189 (VLEWRFDSRL) Sixteen HLA A2+ patients were te in the polytope – one individual rethose 7 recognized more than one-value. | Pol (346–354) VIYQYMDDL Vaccine Vector/Type: DNA prime with vaccinia boost A polyepitope vaccine was generated in a vaccinia construct t HHD mice have a transgene of HLA A2 linked to the transme in the mice. CTL responses to Gag (77–85) SLYNTVATL, Pol (476–484 observed in HIV polytope HHD-vaccinated mice, and these r No CTL immune responses were generated against HLA A2 Nef 180-189 (VLEWRFDSRL) Sixteen HLA A2+ patients were tested for their ability to ma in the polytope – one individual recognized all seven of these those 7 recognized more than one epitope, but they were not s VIYQYMDDL was recognized by 3 of the HLA-A2 patients. | Pol (346–354) VIYQYMDDL. Vaccine Vacc | human (A2) epitopes, all presented by I of H-2D ^d – this transgene is S) KLTPLCVTL, and Nef ccinia boost. 7-166 (PLTFGWCYKL), P stimulation in culture with altures able to recognize at tients; many patients only I | Woodberry1999 H.A A-2. the only MHC molecule expressed (190-198) AFHHVAREL were ol 346-354 (VIYQYMDDL), and the epitopes selected for inclusion least one of the epitopes, and 6 of ad three peptides tested. |
| RT (179–187) | RT (179–187) VIYQYP Keywords escape, immunotherapy The mutation M184V confers resis 1/28 individuals tested produced F VIYQYIDDL, but failed to recogni | KF (179–187) VIYQYMDDL Keywords escape, immunotherapy. The mutation M184V confers resistance to lamivudine, and is in the middle 1/28 individuals tested produced HIV-1 RT-specific CTL that recognized VIYQYIDDL, but failed to recognize the wildtype epitope VIYQYMDDL. This suggests immunotherapy stimulating anti-VIYQYVDDL responses m | RT (179–187) VIYQYMDDL HIV-1 infection human (A2) Schmitt2000 Keywords escape, immunotherapy. • The mutation M184V confers resistance to lamivudine, and is in the middle of the HLA-A2 epitope VIYQYMDDL. • 1/28 individuals tested produced HIV-1 RT-specific CTL that recognized the peptide representing the lamivudine escape mutants VIYQYNDDL and VIYQYIDDL, but failed to recognize the wildtype epitope VIYQYMDDL. • This suggests immunotherapy stimulating anti-VIYQYVDDL responses maybe helpful for reducing lamivudine escape. | human (A2) pitope VIYQYMDDL. enting the lamivudine esca ducing lamivudine escape. | Schmitt2000 upe mutants VIYQYVDDL and |
| RT (179–187) | RT (179–187) • Of 98 patients in cross-patients, respectively) | VIYQYMDDL sectional analysis, 78% had CTL | RT (179–187) VIYQYMDDL HIV-1 infection human (A2) Haas1998 • Of 98 patients in cross-sectional analysis, 78% had CTL against pol – RT was more immunogenic than Integrase and Protease (81%, 51%, and 24% of 37 patients, respectively) | human (A2) genic than Integrase and Pt | Haas1998 otease (81%, 51%, and 24% of 37 |
| RT (179–187) | Pol (339–347 93TH253 VIYQYN subtype CRF01) Keywords HIV exposed persistent Epitope name P334-342. This was a study of HIV-1 exposed HLA-A11 is very common in this and CTL responses were found in 8. | Pol (339–347 93TH253 VIYQYMDDL subtype CRF01) Keywords HIV exposed persistently seronegative (HEPS) Epitope name P334-342. This was a study of HIV-1 exposed persistently seronegative HLA-A11 is very common in this population, and was emand CTL responses were found in 8/8 HIV+ controls, and This epitope was reactive in HIV+ control study subject 14 | Pol (339–347 93TH253 VIYQYMDDL HIV-1 infection human (A2) Sriwanthana2001 subtype CRF01) Keywords HIV exposed persistently seronegative (HEPS). Epitope name P334-342. • This was a study of HIV-1 exposed persistently seronegative (HEPS) female sex workers in Chiang Mai, northern Thailand. • HLA-A11 is very common in this population, and was enriched among the HEPS sexworkers – weak CTL responses were detected in 4/7 HEPS women, and CTL responses were found in 8/8 HIV+ controls, and 0/9 HIV- women that were not exposed. • This epitope was reactive in HIV+ control study subject 144 who carried HLA-A2. | human (A2) hiang Mai, northern Thaila s – weak CTL responses w | Sriwanthana2001 nd. ere detected in 4/7 HEPS women, |
| RT (179–187) | Pol (339–347 93TH253 VIYQYM subtype CRF01) Keywords inter-clade comparisons • More than half of a cohort of HIV epitopes in this group, although E or 2/4 tested FSWs recognized the E or This epitope was conserved in man | Pol (339–347 93TH253 VIYQYMDDL HIV-1 infection subtype CRF01) Keywords inter-clade comparisons. More than half of a cohort of HIV+ female sex workers (FSW) from Northern Thailar epitopes in this group, although E clade versions of previously defined B-clade A2 and A24 tested FSWs recognized the E clade version of this epitope, which is identical to the This epitope was conserved in many subtypes, and exact matches were very uncommon. | Pol (339–347 93TH253 VIYQYMDDL HIV-1 infection human (A2) Bond2001 subtype CRF01) Keywords inter-clade comparisons. More than half of a cohort of HIV+ female sex workers (FSW) from Northern Thailand were HLA-A11 positive, and this study concentrated on A11 epitopes in this group, although E clade versions of previously defined B-clade A2 and A24 epitopes were also tested. 2/4 tested FSWs recognized the E clade version of this epitope, which is identical to the previously defined B clade version VIYQYMDDL. This epitope was conserved in many subtypes, and exact matches were very uncommon. | human (A2) re HLA-A11 positive, and pitopes were also tested. ously defined B clade versi | Bond2001 this study concentrated on A11 on VIYQYMDDL |
| RT (179–187) | RT (179–187) VIYQYMDDL Keywords rate of progression, acute infection. | VIYQYMDDL ession, acute infection. | HIV-1 infection | human (A2) | Day2001 |

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| | The CTL response to optimally defined CTL studied in eight HIV-1-infected subjects, two 2 to 17 epitopes were recognized in a given epitopes were targeted by at least one person. | ptimally defined CTL epitope infected subjects, two with acceptains a given individute at least one person. | The CTL response to optimally defined CTL epitopes restricted by HLA class I A and B alleles in individuals who coexpressed HLA A2, A3, and B7 was studied in eight HIV-1-infected subjects, two with acute infection, five with chronic, and one long-term non-progressor (LTNP) 2 to 17 epitopes were recognized in a given individual, A2-restricted CTL response tended to be narrow and never dominated the response, and 25/27 epitopes were targeted by at least one person. | leles in individuals who coexy e long-term non-progressor (I d to be narrow and never dor | oressed HLA A2, A3, and B7 was TNP) ninated the response, and 25/27 |
| RT (179–187) | Pol (346–354 LAI) VIYQYMDDL Keywords HAART, epitope processing. Ritonavir (RTV) inhibits chymotryptic a there is concern protease inhibitors may relevant concentrations of RTV when the RTV did not alter the presentation two F the MB1 beta subunit, and VIYQYMDI destroyed by MB1 in the constitutive professing and or inhibit the processing and | Reywords HAART, epitope processing. Ritonavir (RTV) inhibits chymotryptic activity in the there is concern protease inhibitors may adversely effevant concentrations of RTV when the proteasome RTV did not alter the presentation two RT A2 epitop the MB1 beta subunit, and VIYQYMDDL which is destroyed by MB1 in the constitutive proteasome. RTV did not inhibit the processing and assembly of | Pol (346–354 LAI) VIYQYMDDL Keywords HAART, epitope processing. Rionavir (RTV) inhibits chymotryptic activity in the 20S proteasome <i>in vitro</i> , as does Saquinavir (SQV) to a lesser extent; Indinavir (IDV) does not. Thus there is concern protease inhibitors may adversely effect CTL epitope processing, but this paper indicates that processing is not inhibited at therapeutically relevant concentrations of RTV when the proteasome is functioning in an intracellular context. RTV did not alter the presentation two RT A2 epitopes processed by distinct pathways: ILKEPVHGV, generated by the constitutive proteasome containing the MB1 beta subunit, and VIYQYMDDL which is dependent on IFNgamma induction of LMP7 which replaces MB1 in the immunoproteasome, and is destroyed by MB1 in the processing and assembly of HLA-B35 or -A2, which are assembled with a rapid and moderate time course, respectively, or of | human (A2) uinavir (SQV) to a lesser externation in the case of t | Kelleher2001a it; Indinavir (IDV) does not. Thus it is not inhibited at therapeutically constitutive proteasome containing in the immunoproteasome, and is time course, respectively, or of |
| RT (179–187) | Pol (334–) Reywords binding affir Epitope name Pol334. Assay type CD8 T-cell HLA-A2-restricted HIV mice, and responses to trecognized per patient. This epitope was one of | Pol (334–) NIYQYMDDL HIV-1 infection Keywords binding affinity, inter-clade comparisons, computational epitope Epitope name Pol334. Assay type CD8 T-cell Elispot - IFN \(\text{Thromium-release} assay, \text{Flow cyton} \) HLA-A2-restricted HIV-1 CTL epitopes were computationally predicted. B mice, and responses to the peptides in 17 HIV-1 infected patients were tested recognized per patient. This epitope was one of the previously identified HLA-A2 epitopes studied. 1/17 HIV-infected HLA-A2+ people in this study recognized this epitope. | Pol (334–) VIYQYMDDL HIV-1 infection human (A2) Corbet2003 Keywords binding affinity. inter-clade comparisons, computational epitope prediction. Epitope name Pol334. Assay type CD8 T-cell Elispot - IFNY, Chromium-release assay, Flow cytometric CTL assay. HLA-A2-restricted HIV-1 CTL epitopes were computationally predicted. Binding affinities for HLA-A**0204, immunogenicity in HLA-A**0201 transgenic mice, and responses to the peptides in 17 HIV-1 infected patients were tested. 31 novel conserved A2 epitopes were detected. An average of 4 epitopes were recognized per patient. • This epitope was one of the previously identified HLA-A2 epitopes studied. • I/17 HIV-infected HLA-A2+ people in this study recognized this epitope. | human (A2) y. for HLA-A*0204, immunoge.erved A2 epitopes were detec | Corbet2003 snicity in HLA-A*0201 transgenic ted. An average of 4 epitopes were |
| RT (179–187) | Pol (subtype B) VIYQYMM Keywords inter-clade comparisons. HIV-specific CTL were found in exp. Seroprevalence in this cohort is 90-9 Most isolated HIV strains are clade responses are frequently observed us. This epitope is conserved among A, | Pol (subtype B) VIYQYMMDL HIV-1 exposed Keywords inter-clade comparisons. HIV-specific CTL were found in exposed seronegative prostitutes from Na Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is amon, Most isolated HIV strains are clade A in Nairobi, although clades C and responses are frequently observed using A or D clade versions of epitopes. This epitope is conserved among A, B and D clade viruses. | Pol (subtype B) VIYQYMMDL HIV-1 exposed seronegative human (A2, A*0202) Rowland-Jones1998b Keywords inter-clade comparisons. • HIV-specific CTL were found in exposed seronegative prostitutes from Nairobi – these CTL may confer protection. • Seroprevalence in this cohort is 90-95% and their HIV-1 exposure is among the highest in the world. • Most isolated HIV strains are clade A in Nairobi, although clades C and D are also found – B clade epitopes are often cross-reactive, however stronger responses are frequently observed using A or D clade versions of epitopes. • This epitope is conserved among A, B and D clade viruses. | human (A2, A*0202) L may confer protection. he world. – B clade epitopes are often | Rowland-Jones1998b cross-reactive, however stronger |
| RT (179–187) | RT (346–354 LAI) VIY Vaccine Vector/Type: peptide Keywords binding affinity, vac Epitope name LR26. The stability of peptide binding (p17), SLLNATDIAV (gp41) & RGPGRAFVTI and VIYQYM rhan an hour. | VIYQYMDDL eptide Strain: B clade LAI nity, vaccine-specific epitope of binding to HLA-A2.1 was det gp41) and LLWKGEGAV (RYQYMDDL bound with a low eptides formed stable complex | RT (346–354 LAI) VIYQYMDDL Vaccine mouse (A2.1) Peter2001 Vaccine Vector/Type: peptide Strain: B clade LAI Adjuvant: Incomplete Freund's Adjuvant (IFA), Montanide (ISA 720), P30, PLG Keywords binding affinity. vaccine-specific epitope characteristics, immunodominance. Epitope name LR26. The stability of peptide binding to HLA-A2.1 was determined for six HLA-A2.1 peptides included in this vaccine study – ILKEPVHGV (RT), SLYNTVATL (p17), SLLNATDIAV (gp41) and LLWKGEGAV (RT) all bound with high affinity comparable to a influenza epitope reference (GILGFVFTL), while RGPGRAFVTI and VIYQYMDDL bound with a lower affinity (relative binding activity = 0.01). The four high-affinity peptides formed stable complexes with half-lives ranging between 8 and 32 hours, while the low affinity peptides had half lives of less than an hour. | mouse (A2.1) vant (IFA), Montanide (ISA 7 cluded in this vaccine study – arable to a influenza epitope 1 0.01). and 32 hours, while the low aff | Peter2001 20), P30, PLG ILKEPVHGV (RT), SLYNTVATL :eference (GILGFVFTL), while inity peptides had half lives of less |

| HXB2 Location | Author's Location | Sequence | Immunogen | Species (HLA) | References |
|---------------|---|---|---|--|--|
| | HLA-A2.1 transgenic as adjuvants. All peptides except VJ alone, indicating immu | HLA-A2.1 transgenic mice were immunized with the six HIV-1 pepti as adjuvants. All peptides except VIYQYMDDL induced a stong CTL response in alone, indicating immunodominance when the combination was used. | HLA-A2.1 transgenic mice were immunized with the six HIV-1 peptides and P30, as a universal T-helper epitope, with IFA or Montanide or microspheres as adjuvants. All peptides except VIYQYMDDL induced a stong CTL response in Cr-release assays - stronger responses were observed when peptides were delivered alone, indicating immunodominance when the combination was used. | versal T-helper epitope, with tronger responses were obser | IFA or Montanide or microspheres wed when peptides were delivered |
| RT (179–187) | RT (346–354 LAI) VIX Vaccine Vector/Type: peptide Keywords vaccine-specific epi Epitope name LR26. • When HIV-1 peptides were use given individually, but immune counteract immunodominance HLA-A2.1-epitope CTL respon in the spleen. | RT (346–354 LAI) VIYQYMDDL Vaccine Vaccine Vector/Type: peptide Strain: B clade LAI Adjuvant: Incom Keywords vaccine-specific epitope characteristics, immunodominance. Epitope name LR26. When HIV-1 peptides were used to vaccinate HLA-A2.1 transgenic A2 given individually, but immunodominance limited the response to son counteract immunodominance in BALB/c mice, so it was given with tH.A-A2.1-epitope CTL responses, but not Kb CTL responses. This wiin the spleen. | RT (346–354 LAI) VIYQYMDDL Vaccine Tream Toomplete Freund's Adjuvant (IFA), IL-12, P30 Vaccine Vector/Type: peptide Strain: B clade LAI Adjuvant: Incomplete Freund's Adjuvant (IFA), IL-12, P30 Keywords vaccine-specific epitope characteristics, immunodominance. Epitope name LR26. • When HIV-1 peptides were used to vaccinate HLA-A2.1 transgenic A2-Kb mice, strong responses to five peptides were observed when the peptides were given individually, but immunodominance limited the response to some of the peptides when they were given in combination [Peter2001]. IL-12 can counteract immunodominance in BALB/c mice, so it was given with the multiple epitope vaccination, and was instead found to specifically eliminate the HLA-A2.1-epitope CTL responses, but not Kb CTL responses. This was possibly a consequence of transient depletion of T-cells, B cells and macropahges in the spleen. | mouse (A2.1) tvant (IFA), IL-12, P30 sponses to five peptides were when they were given in com vaccination, and was instead quence of transient depletion of | Peter2002 e observed when the peptides were bination [Peter2001]. IL-12 can found to specifically eliminate the of T-cells, B cells and macropahges |
| RT (180–189) | RT (LAI) Recognized by CTL fr A previous study deter | RT (LAI) IYQYMDDLYV HIV-1 infection Recognized by CTL from a progressor, spans important RT functional domain. A previous study determined that this was an epitope recognized by a long-term | RT (LAI) IYQYMDDLYV HIV-1 infection Recognized by CTL from a progressor, spans important RT functional domain. A previous study determined that this was an epitope recognized by a long-term survivor. | human (A*0201) | Menendez-Arias1998, vanderBurg1997 |
| RT (181–189) | RT (181–189 LAI) Keywords binding aff This epitope contains 1 High levels of recogni patient 250#0 (HLA-A Both the wild-type and | RT (181–189 LAI) YQYMDDLYV Keywords binding affinity, computational epitope prediction. This epitope contains the mutation M184V, a frequent mutatic High levels of recognition by ELISPOT were observed for zi patient 250#0 (HLA-A*0201), but neither were recognized by Both the wild-type and the mutated peptide were computer pr | RT (181–189 LAI) YQYMDDLYV HIV-1 infection human (A*0201) Samri2000 Keywords binding affinity, computational epitope prediction. This epitope contains the mutation M184V, a frequent mutation induced by nucleoside reverse transcriptase inhibitors. High levels of recognition by ELISPOT were observed for zidovudine induced mutation YQYVDDLYV and for the wildtype peptide YQYMDDLYV in patient 250#0 (HLA-A*0201), but neither were recognized by patient 201#5 (also HLA-A*0201) Both the wild-type and the mutated peptide were computer predicted to have a high binding affinity for A2 (http//bimas.dcrt.nih.gov/molbio/hla_bind) | human (A*0201) rse transcriptase inhibitors. (QYVDDLYV and for the with the following) gaffinity for A2 (http//bimas. | Samri2000 ildtype peptide YQYMDDLYV in dcrt.nih.gov/molbio/hla_bind) |
| RT (192–201) | RT (192–201) • Of 98 patients in cross patients, respectively) • New clusters of epitop | RT (192–201) DLEIGQHRTK HIV-1 infection of 98 patients in cross-sectional analysis, 78% had CTL against pol – RT patients, respectively) New clusters of epitopes were defined utilizing different HLA molecules. | RT (192–201) DLEIGQHRTK HIV-1 infection human (A3) Haas1998 • Of 98 patients in cross-sectional analysis, 78% had CTL against pol – RT was more immunogenic than Integrase and Protease (81%, 51%, and 24% of 37 patients, respectively) • New clusters of epitopes were defined utilizing different HLA molecules. | human (A3) nogenic than Integrase and P | Haas1998 rotease (81%, 51%, and 24% of 37 |
| RT (192-216) | RT (359–383 HXB2) DLEIGG RWGLT' | DLEIGQHRTKIEELRQHLL— HIV-1 infe RWGLTT efined for RT-specific CTL clones in this study. | .– HIV-1 infection s in this study. | human (Bw60) | Menendez-Arias1998, Walker1989 |
| RT (192–216) | RT (191–215) B RY Keywords HAART, escape. • Polyclonal CTL recognition variant, RT T215Y. | DLEIGQHRTKIEELRQHLL- RWGFTT scape. gnition switched from RT 191-215 t | RT (191–215) DLEIGQHRTKIEELRQHLL- HIV-1 infection human (polyclonal) Haas1997, Menendez-Arias1998 Keywords HAART, escape. Polyclonal CTL recognition switched from RT 191-215 to RT 514-524 when AZT therapy selected for the resistance mutation, and presumably the escape variant, RT T215Y. | human (polyclonal) selected for the resistance m | Haas1997, Menendez-Arias1998 utation, and presumably the escape |
| RT (198–212) | RT (SF2) • This epitope was mappe an HLA-B60 individual | HRTKIEELRQHLLRW ped by ELISPOT in a study ident al. | RT (SF2) HRTKIEELRQHLLRW HIV-1 infection human Altfeld2000b • This epitope was mapped by ELISPOT in a study identifying new HLA-B60 epitopes, and was one of the epitopes presented by another HLA molecule in an HLA-B60 individual. | human was one of the epitopes pres | Altfeld2000b ented by another HLA molecule in |